The MR of the brainstem showed a small ischaemic lesion in the left dorsolateral medulla without any further pathology.

Until confirmation of the total occlusion of the left vertebral artery the patient was treated with cumarine to prevent arterial embolism. The clinical symptoms progressively disappeared over the next 30 days and the patient left hospital with only a slight ataxic gait and mild dysaesthesia within the left C6 segment.

Discussion

The patient presented with an anterior displacement and fracture of the left facet joint between level C5 and C6 and radicular pain in the left C6 distribution, and subsequently developed a left vertebral artery dissection with left brainstem ischaemia. It is suspected that the dissection occurred during the 19 day delay with subsequent occlusion including the posterior infracerebellar artery. By contrast with our case, most reports during the past decade concerning posterior circulation ischaemia connected with vertebral artery occlusion occurred spontaneously or were loosely related to minor trauma during sport or neck manipulation during chiropraxy. These typically occurred in young or middle aged adults, equally between the sexes.¹ Although traumatic fractures of the cervical spine occur often, injuries of the vertebral arteries with or without clinical symptoms are rarely evident. The incidence is postulated to be between 3% and 10%.²

In a retrospective study Parent and coworkers reviewed some 640 patients sustaining fractures of the cervical spine. Of these, 96 had facet involvement and in only five was injury of the vertebral artery diagnosed by initial major neurological deficits such as cerebellar infarction, cortical blindness, or pontine infarction, which have been documented by postmortem examination in two cases.³ All these patients had cervical fractures located at C5-C6 and in one case in combination with a fracture at C6-C7. Radiographs showed anterior dislocation at C5-C6 in four cases and at C6-C7 in one case. Bilateral facet fractures were evident in four cases.

Only one prospective study exists that considers the combination of facet joint dislocation of the cervical spine, the incidence of vertebral artery injury, and the neurological deficit.⁴ From 12 consecutively examined patients with facet joint dislocation (C5/C6 in seven, C6/C7 in three, and C4/C5 in two) nine showed an occlusion of at least one vertebral artery. Of these nine patients only two with bilateral facet joint dislocations had a transient neurological deficit. Further indications of a traumatic or spontaneous dissection of the vertebral artery are neck pain and symptoms of a C6 root compression.⁵

Thus a combination of neurological and radiological findings could lead to an early diagnosis and may indicate development of a dissection of the vertebral artery. We suggest that patients with the clinical symptoms and type of injury described here are prone to development of a dissection or occlusion of the vertebral artery. Early diagnostic procedures by non-invasive diagnostic techniques such as MR angiography and ultrasound techniques coupled with treatment at the onset of a possible dissection may help to prevent the formation of a microembolism or arterial occlusion.

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Herpes simplex virus type 2 ascending myeloradiculitis: MRI findings and rapid diagnosis by the polymerase chain method

Although neurotropic viruses are often suspected of causing spinal cord injuries, confirmation by early diagnosis is difficult. Ascending myelitis related to herpes simplex virus type 2 (HSV-2) infection has seldom been reported and the diagnosis could be established only at postmortem examination.¹⁻⁵ We report the case of an elderly woman with a subacute ascending myeloradiculitis. MRI showed spinal cord and sacral root involvement and the polymerase chain reaction allowed the rapid identification of HSV-2 DNA in the CSF.

A 76 year old woman was referred because of urinary retention and paraparesis. Three weeks previously, she had noted the progressive onset of anorexia, fever (38°C), weight loss (4 kg), and low back pain. Evaluation performed in another hospital showed negative bacterial cultures from blood and urine and CT of the thorax, abdomen, and lumbosacral spine was normal. Three days before admission she complained of right sciatalgia and rapidly developed lower extremity weakness and sphincter disturbances.

Neurological examination showed a flaccid paraplegia, a T10 sensory level, and a distended bladder. Deep tendon reflexes were absent in the lower limbs and plantar responses were both extensor. In the upper extremities strength was normal but reflexes were brisk and a bilateral Hoffman sign was noted. Mental state and cranial nerves were normal.

Non-enhanced T1 weighted images of the spine were normal. T2 weighted sequences showed a linear hyperintense signal at the T10 level and within the conus medullaris. T1 weighted images with contrast injection showed an enhancement of both the posterior meninges and the roots of the cauda equina.

Her CSF contained 73 leucocytes/mm³ (97% lymphocytes), 132 mg/dl protein and 68 mg/dl glucose. Electrophoresis of CSF protein showed 26% γ -globulins with an oligoclonal distribution and a raised IgG/albumin ratio (0.56; normal <0.25). A polymerase chain reaction was performed on CSF with a pair of primers that allowed the simultaneous detection of four viruses

of the herpes group. A strong signal was obtained on ethidium bromide stained gel. Characterisation of HSV-2 DNA was achieved by restriction analysis of the amplified product. α -Interferon in CSF was normal. The patient had no history of recurrent herpes genitalis. There was no serological evidence for borreliosis, HIV-1 or HIV-2, HTLV-I, Q-fever, listeriosis, cytomegalovirus, measles, varicella zoster, or Epstein-Barr virus infection. CD4 counts were normal and no cause for immunodepression could be identified.

Parenteral acyclovir (30 mg/kg daily) was given for 10 days and the patient's neurological state remained unchanged. Six days after admission, sparse vesicular lesions appeared on the patient's buttocks, internal aspects of the thighs, and lower part of the abdomen. Ten days later, numbness in both hands appeared. Examination showed bilateral arm and shoulder weakness and the disappearance of upper limb reflexes. The patient then became confused and drowsy, developed hyponatraemia and hyperazotaemia and died on day 21 after admission. Necropsy examination was not performed. Subsequently, CSF cultures were reported as positive for HSV-2. Analysis of serial serum samples showed a sevenfold rise in anti-HSV antibodies between admission and death. IgM antibodies were detected in one early serum sample. Analysis of the CSF and serum anti-HSV antibodies ratio showed the existence of specific intrathecal synthesis.

Various clinical syndromes have been linked to HSV-2 involvement in the nervous system. HSV-2 encephalitis typically occurs in the newborn, but accounts for less than 5% of herpetic encephalitis in children and adults. Acute, self limited meningitis is found in young adults with primary genital HSV infection. Sacral radiculitis with perineal dysaesthesias, autonomic dysfunction, and sometimes mild lower limb weakness may also be associated with herpes genitalis in young adults. In most cases, neurological symptoms occur two to seven days after the genital eruption and patients recover within three weeks.

By contrast, HSV-2 involvement in the spinal cord is rare and severe. Two cases of HSV-2 extensive myeloradiculitis have been reported in patients with AIDS simultaneously infected with cytomegalovirus,^{1,2} and two others in diabetic patients.^{3,4} HSV-2 necrotising myelopathy has also been found in association with malignancy.⁵ In all cases the outcome was fatal within four to seven weeks, and the diagnosis could only be made at necropsy, when HSV-2 was recovered from the spinal cord. There has been a single report of HSV-2 myelitis with a favourable outcome in which the virus was isolated from the CSF.⁶

In our patient, MRI clearly showed myeloradiculitis, on T2 weighted and gadolinium enhanced T1 weighted sagittal sequences. Although well correlated with the clinical features, however, MRI findings lacked specificity.

The direct diagnosis of nervous system infection by HSV is difficult as isolation of the virus from CSF is most often unsuccessful and serological confirmation is too late. Recently, the polymerase chain reaction has proved to be a powerful tool in the rapid diagnosis of meningoencephalitis due to herpes viruses. In this case, it allowed us to identify HSV-2 in the CSF immediately

after admission, a week before the appearance of the cutaneous eruption.

The pathogenesis of HSV-2 myelitis in this case remains ill defined. Although IgM antibodies were detected in the first serum sample we are not sure that the patient's myeloradiculitis reflected true HSV-2 primary infection because reappearance of IgM antibodies can be detected in herpes virus reactivation. All but one previously reported case of HSV-2 myelitis were fatal and occurred in immunocompromised hosts. This patient was neither diabetic nor HIV-infected, but despite normal CT examinations of the thorax and abdomen, a hidden malignancy cannot be excluded. Reactivation of a latent infection within dorsal root ganglia neurons with a contiguous spread via sacral ganglia to the spinal cord has already been proposed in HSV-2 ascending myelitis.³ It is of note that despite antiviral treatment, our patient died within six weeks. Thus an immunologically mediated injury triggered by the herpes infection could also be involved here, as in another patient with HSV-1 myelitis whose necropsy examination showed patchy demyelination of the spinal cord.⁷

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Peduncular hallucinosis and right hemiparkinsonism caused by left mesencephalic infarction

Since the original description of Lhermitte¹ several causes of peduncular hallucinosis have been reported, but always in relation to a bilateral mesencephalic lesion. We describe here a patient with prolonged visual hallucinations and right hemiparkinson-

ism. An MRI showed a unilateral infarction involving the left cerebral peduncle.

A previously healthy 70 year old right handed woman presented with visual hallucinations. Two months previously, she began, one night, to see objects (motorbikes), animals (dogs, horses), and people (Japanese) entering and driving silently round her room, across the entire visual field. Although the images were of normal colours and sizes, she was aware that they were not real, and never described "déjà vu" or "jamais vu" phenomena, tactile, or auditory hallucinosis. The hallucinatory events became progressively longer and more frequent, lasting from minutes to hours, during both day and night. Her medical history included mild hypertension, normofunctional multinodular goitre, and surgery for left breast carcinoma six years earlier, without subsequent evidence of recurrence. There were no other remarkable personal or familial antecedents. On examination, she was alert, oriented, and cooperative. She remembered four of five words after five minutes, and the mini mental state test was 32/35. She remained in a left tilted posture, and showed severe impairment of postural reflexes, mild bradykinesia, cogwheel rigidity, and intermittent resting tremor in the right extremities (mainly in the lower limb). Tendon reflexes were brisk and increased on the right side, but there was no clonus and the plantar responses were flexor. There were no other remarkable findings on general or neurological examination. Laboratory investigations, including ESR, routine haematological, biochemical, and immunological studies, thyroid function tests, serological tests for syphilis, cerebrospinal fluid examination, EEG, and cranial CT were normal or negative. An MRI showed an abnormal high intensity signal in the left cerebral peduncle on T2-weighted images (figure). Multiple foci of T2-weighted high signal intensity were also seen throughout the periventricular white matter. Such findings were consistent with ischaemic damage. Despite treatment with haloperidol and phenytoin the patient became more impaired within the next several weeks, showing continuous visual hallucinations, with frequent episodes of agitation and disorientation.

Vascular lesion of the upper brainstem is the most often cause of peduncular hallucinosis. A case of peduncular hallucinosis due to bilateral mesencephalic infarction diagnosed by MRI has been recently reported.² The present one is the first report, however, to our knowledge, of peduncular halluci-

Axial T2-weighted MR image obtained with a 0.5-T unit shows an area of hyperintensity in the left cerebral peduncle, consistent with infarction.

nosis due to unilateral lesion. Although the precise anatomical basis for peduncular hallucinosis remains unclear, it seems that the substantia nigra pars reticulata (SNpr) is directly implicated.³ Little is known about the pathogenesis of peduncular hallucinosis. A relation with the rapid eye movement (REM) phase of sleep has been proposed. In this sense, it is known that the SNpr may play an important part in the regulation of the different phases of sleep through its connections with centromedian/parafascicular nuclei of the thalamus, superior colliculus, and reticular formation.⁴

Finally, although we cannot rule out the presence of brainstem Lewy bodies, it is probable that the right hemiparkinsonism found in our patient was related to ischaemic damage of the left substantia nigra pars compacta.⁵

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Intraneural ganglion of the sciatic nerve: detection by ultrasound

Intraneural ganglia are a rare cause of peripheral nerve lesions most often affecting the peroneal nerve. Their origin is unknown. Some 50 cases were reported up to 1979.¹ Sciatic nerve ganglia are very rare. We report a sciatic nerve lesion caused by a giant ganglion situated at the level of the distal thigh and damaging the tibial portion only.

A 36 year old male right handed and right footed state officer complained of pain in his right calf for about six years especially when jogging and walking for more than 30 minutes. He was treated for a lumbar disc hernia, but a lumbar CT was unremarkable. Simultaneously he noted discomfort in his right toes when wearing shoes; this was relieved by wearing orthopaedic sandals.

On first neurological examination he was found to have Lasègue's sign with his right leg at an angle of about 85°. Reflexes and sensation in the lower extremities were normal. There was moderate paresis of the toe flexors. Plain radiographs of the lower spine showed six lumbar vertebrae and a fissured vertebral arch of the first sacral segment.